Clinical Section

Prevention of Pulmonary Embolus

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This paper will demonstrate some of the recent advances in diseases of veins and also attempt to point out that in most cases, pulmonary embolism can no longer be regarded as an unforeseen and unpreventable accident.

It is axiomatic that to develop pulmonary embolus, there must be associated peripheral thrombophlebitic process. In many types of thrombophlebitis, there is a recognized active surgical treatment, for instance in:

(1) Thrombophlebitis of the angular vein in association with infections of the face; one ligates and divides the angular vein to prevent cavernous sinus thrombosis.

(2) Thrombophlebitis of the lateral sinus; one ligates and divides the jugular vein.

(3) Appendicitis with chills usually means portal pyaemia. The organism is usually a streptococcus, and the only effective treatment is a ligation and division of the ileo-colic vein before it enters the right colic. (Braun, 1913.)

It is interesting to note that in spite of the extreme commonness of suppurative adenitis in the neck or deep infections of the neck and jaw, that one rarely sees Thrombophlebitis of the jugular vein. In fact it is only seen when it spreads from a thrombosed lateral sinus which slows the jugular current. I would like to suggest that the cause of this infrequent involvement is due to the rapid current in the jugular vein which is caused by (1) large blood volume; (2) the erect posture, and (3) the direct action of thoracic suction.

A similar absence of pulmonary embolus is noted in other examples of Thrombophlebitis, notable in that type associated with varicose veins. Here again the diseased vein wall favors firm attachment of the clot, and the saphenofemoral anastomosis presents a rapid current in the femoral vein which halts the propagation of the clot. Naturally, this rapid femoral current is absent in the post-operative bedridden patient so that it is possible to develop embolism postoperatively from Thrombophlebitis in varicose veins.

It is unnecessary to go into the cause or causes of Thrombophlebitis. In many instances, contiguity with infective processes plays a large rôle. Viz: Thrombophlebitis of angular vein or lateral sinus. In other instances, trauma plays a part, then there are possible changes in the blood elements playing a rôle in coagulation. But certainly, one of the most obvious factors, as pointed out, is slowing of the venous current, and it is even more obviously a factor in extension of the Thrombophlebitic process. The stasis has many causes: (1) Dehydration, (2) abdominal distension, (3) reclining posture, and (4) immobilization of legs and diaphragm, etc.

A thrombus once formed tends to propagate downstream, i.e., in the direction of the venous current, reaching larger and larger channels and becoming less and less well attached until it eventually reaches a channel which is much too large and continuing to propagate waves idly in the slow stream, eventually to fracture and become an embolus. This propagation of the thrombus is the dangerous feature and can be prevented by speeding up the venous circulation. This is what happens in the average case of varicose thrombophlebitis, the process being arrested at the femoral vein where the current is rapid. Except post-operatively, these patients rarely develop embolism. Therefore, there are three obvious methods of preventing pulmonary embolism.

Methods of Prevention

1. Prevent thrombosis by preventing a slow current or by the use of heparin or coumarin. It might be pointed out here that the Trendelenberg position favours a rapid current below the diaphragm and is used routinely by Wangensteen for ten days post-operatively, and that, on the other hand, the semi-reclining position with flexed knees caused by the Gatch bed, although comfortable, has probably directly caused many cases of pulmonary embolus.

2. Prevent propagation of the clot by the interposition of a rapid current (Trendelenberg position) or by the use of heparin or coumarin.

3. Prevent the embolus from reaching the right heart by blocking its path by ligation and division of the vein above the thrombus.

(NOTE: Heparin and Coumarin neutralize the effect of both prothrombin and thrombin. Heparin is very expensive, $5.20 for 100 mgms with average requirements of 2-3 hundred mgms daily for 3-10 days. Coumarin is effective and the action is prolonged and tends to be cumulative, and the dosage is not standardized, so that for immediate emergency effect, it is not practical. Both drugs are not without danger from haemorrhage. I have had two very serious, almost fatal, post-operative haemorrhages from the use of coumarin. It is important to remember that the effect of coumarin progresses in many cases after the drug is discontinued.)

To carry out this preventative treatment, it is necessary that we be able to recognize this thrombophlebitic process. The following is a classification of thrombophlebitis excluding of course those types previously mentioned in contiguity with an infective process.

A. Superficial
1. thrombophlebitis in varicose veins;
2. thrombophlebitis in non-varicose veins (phlebitis migrans)
   a. ideopathic
   b. Buegers
   c. cachexia

B. Deep—

1. Effort thrombophlebitis of axillary vein.
2. Thrombophlebitis of prostatic and uterine veins.
3. Femoro-iliac thrombophlebitis (milk leg).
4. Mesenteric thrombophlebitis.
5. Deep peripheral thrombophlebitis (deep veins of calf).

I shall now briefly review the above varieties of thrombophlebitis, point out the treatment and indicate the relationship of each to pulmonary embolus.

1. **Thrombophlebitis in varicose veins**—usually starts at the level of the knee and naturally at the medial aspect. It travels upwards in a slow erratic fashion and once half way to the groin, it usually goes the rest of the way to the sapheno-femoral anastomosis but rarely causes embolism, because at the femoral vein it meets a rapid current which stops propagation, and the previously unhealthy vein wall favours firm attachment of the thrombus. Post-operatively, there is no rapid current in the femoral vein, so although it is rare, it does happen that this variety of thrombophlebitis can and does give rise to pulmonary embolism and is the source of the embolus in 2%-6% of cases. Naturally, the treatment of this type of embolus is pre-operative ligation and division of saphenous vein at its junction with the femoral.

There are two ways to make this disease last a long time, either of which is usually done.

1. Go without an elastoplast.

2. Go to bed in semi-sitting position. Both keep the stream slow and encourage the process. So when it occurs in the lower leg, use elastoplast and ambulatory treatment, and when it occurs in the upper leg, have the patient go to bed in the Trendelenberg position, or better still, in all cases, no treatment can compare with early division and ligation of the saphenous vein at its origin for speed of recovery and prevention of recurrence.

2. **Thrombophlebitis in non-varicose superficial veins (Phlebitis Migrans)**, is a common accompaniment of Buerger's disease and often points to the diagnosis, but is also seen in the cachetic state. Aside from these types, there is a large group in which the disease is apparently idiopathic. There is a tendency to recurrence throughout life when an inch or two of superficial vein usually in the lower leg becomes thrombophlebitic for a week or two and disappears spontaneously, only to recur elsewhere after a varying period of time. There is no tendency to pulmonary embolism in this disease and ordinarily, ambulatory treatment with supporting dressing of elastoplast or Unna's paste is satisfactory. If the saphenous veins can be demonstrated to be in any way incompetent, it is wise to divide and ligate the saphenous at its origin.

3. **Effort Thrombophlebitis of the axillary vein**—is rather unusual. I have seen two cases. It usually occurs in the right arm of active males in the 3rd, 4th and 5th decades. The cause is not known, but may be squeezing of the vein between the subclavius muscle and first rib. The arm is painful, swollen and cyanotic with dilated superficial veins, and usually recovers in ten days of rest and elevation on pillows. Occasionally, these patients develop permanent peripheral vasospastic phenomena, indicated by parathesias, coldness and pain with chronic oedema. It is reflex in origin and can be cured by a venectomy, a cervical sympathectomy or by novocainization of cervical sympathetics. This disease does not give rise to embolus because the axillary current always meets the rapid current of the jugular which prevents propagation of the clot.

4. **Thrombophlebitis of Prostatic and (or) uterine veins.** This pelvic variety was once thought to be the most common origin of pulmonary embolus. There is no way of diagnosing this condition during life unless an embolus can be proved to have no other source. It only accounts for 6% of emboli. If suspected, the patient is put in immediate Trendelenberg position and since no further active treatment is possible and because the progress of the disease cannot be observed, its suspicion is a positive indication for heparin or coumarin.

When this condition is suspected and during its treatment, the more common origins of emboli must be repeatedly examined (as will later be described) because this is certainly a relatively unlikely source.

5. **Femoro-iliac Thrombophlebitis (Phlegmasia alba dolens or milk leg).** This condition is twice as common after pelvic operation and is said to be commoner in the left leg, although not in my experience. It also occurs after childbirth or spontaneously. Usually the whole lower limb is swollen, but the absence of oedema is no proof the disease does not exist. The swelling is apparently due to venospasm, reflex in origin, and not to venous or lymphatic obstruction—proved by the fact that Oschener and De Bahey, Leriche, Homans and others have been able permanently to relieve the oedema by novocainization

of the homolateral lumbar sympathetic ganglia. It is initiated by pain in the groin, occasionally severe and rarely very severe, resembling arterial embolus due to terrific reflex vasospasm. It is often preceded by elevation of temperature and pulse. The local pulse is often diminished or absent (vasospasm). The leg is obviously large and white with varying degrees of tenderness along course of femoral vein and, when erect, the foot becomes dusky and the superficial vein tends to become engorged. The involved leg is usually noticeably cooler than the normal one due to vasospasm. This disease has interesting sequelae.

1. Rarely gives rise to pulmonary embolism, contrary to popular opinion, and only accounts for 2% of pulmonary emboli.
2. The oedema and vasospastic phenomena usually clear up surprisingly well contrary to popular opinion.
3. They may develop compensatory varicosities first in the upper outer thigh and not along the usual saphenous distribution.
4. May give rise to post-thrombophlebitic induration and ulceration, diagnosed by the history of milk leg, the atypical varicosities and the typical herald patch of induration. This type of ulceration will not respond to the usual treatment of varicose ulcer.

The treatment of milk leg is (1) to put the patient in Trendelenberg position to hasten the current and stop propagation; (2) to encourage leg exercises and massage for the same reason. If the patient develops a non-fatal embolus (which is rare), one must decide between heparin or coumarin and ligation and section of the external iliac vein. According to Homans this reduces the incidence of sequelae and absolutely prevents embolism. However, it is usually necessary to do nothing except Trendelenberg position and leg exercises as the disease is essentially not serious.

I recall a case of milk leg in a man in which the origin was apparently idiopathic in whom novocainization of the lumbar sympathetics failed to relieve permanently the pain, oedema and vasospastic phenomena. On exploring his femoral triangle, 6 inches of thrombosed saphenous vein was resected and 3 inches of thrombosed femoral vein also resected. The femoral artery was firmly bound to the femoral vein by a peculiar juicy inflammatory reaction and the artery showed no visible pulsation. As soon as the femoral vein was separated and resected, the artery pulsated normally, even distal to the area of venous resection, indicating that the vasospasm was reflex, the afferent part of the arc likely being peri-venous. The previously cold painful limb became immediately pink and the pain never recurred. Within three days, the leg was normal in size and when last seen, one month post-operatively, there were no signs of recurrence.

6. **Mesenteric Thrombophlebitis.** Most cases have an infectious origin as in appendiceal thrombophlebitis and here the treatment has been indicated. Other cases are associated with mesenteric embolism, and a few are manifestations of Buerger's disease. Naturally, this disease cannot give rise to pulmonary embolism as it is portal in its origin.

7. **Deep peripheral thrombophlebitis (deep veins of the calf).** This is a treacherous, quiet disease, little described and by many not recognized as an entity, yet it has ben proven beyond doubt to be the commonest origin of pulmonary emboli, giving rise to 85% cases. French anatomists were the first to demonstrate the large plexus of veins between the gastrocnemius and soleus and deep to the soleus. It was first pointed out in Vienna that post mortem examination showed this was the commonest embolic origin and it was Homans who popularized the disease in this country.

It occurs post-operatively as does milk leg, but also after trivial injuries below the knee and also during medical diseases which require bed rest.

The disease starts in the calf or less commonly in the sole of the foot and ascends to the calf, and seeing it is the cause of 85% of emboli, the common impression that pulmonary embolus is unavoidable is no longer tenable, because the disease can be recognized before embolus in 75% of cases, and furthermore, seeing that only 1/3 of emboli are fatal, those that have non-fatal emboli with no localizing signs must have the disease in the calf in 85% of cases.

Is the disease worth treating where recognized? The following facts convince me that it is:

1. 6% of the surgical deaths at Rochester are due to pulmonary embolus.
2. 1 person in 17 with deep peripheral thrombophlebitis develops fatal pulmonary embolus.
3. 1 person in 6 who has a non-fatal embolus subsequently dies of fatal embolism.
4. If all fatal emboli preceded by non-fatal emboli could be prevented, then the incidence of death due to embolism could be reduced 66% and the origin in most cases, if varicose, femoroiliac and prostatic or uterine thrombophlebitis can be ruled out, is in the deep veins of the calf.

Assuming the disease is well worth treating, how is it recognized? By repeated examination of the lower leg for the earliest manifestations, which are:

1. Pain or tenderness (often mild) in the sole of the foot, behind medial malleolus or in calf.
2. A dull pain or ache high up in the calf on forced dorsiflexion of the foot (as Homans first described this sign, I have been calling it Homans' sign).

3. Occasional slight fever.
4. Increased heat or coldness (variable) usually colder in my experience.
5. Oedema of ankle (minimal) and in my experience very unusual.
6. Slight cyanosis, noticed as a rule only on dependency.

Certainly, it is a silent disease and must be looked for. Any leg complaints of post-operative patients must never be overlooked, and if an embolus has already occurred, the search must be rigorous. Failure to elicit signs of the disease does not exclude it; this failure may indicate a prostatic or uterine source of the embolus or that the disease is excessively silent. It is important to recognize the disease early as early treatment gives much more satisfactory results.

Treatment of Deep peripheral Thrombophlebitis—depends on whether embolism has occurred or not.

1. BEFORE EMBOLISM

   a. Heparin if practical and available is ideal.
   b. Coumarin gives results but is slow and unpredictable and must be used in conjunction with Homans treatment.
   c. Homans Treatment–immediate Trendelenberg with bed clothes' cradle and no pillow behind knees, with leg and foot exercises and massage for 10 days or until Homans sign disappears for 3 days. Then a level bed for 2 days, then elastoplast strapping and allow the patient up. If Homans sign reappears at any time, the treatment is re-started and if it reappears a third time or if embolism occurs during this treatment, or if the thrombophlebitic process is ascending to thigh, then operation is indicated.
2. AFTER EMBOLISM — Immediate division and ligation of the femoral vein distal to the profunda if not involved. If the profunda is involved, the common femoral must be ligated. This operation causes immediate cessation of all symptoms, reduces the sequelae and permanently eliminates the chance of embolus.

It must not be forgotten that the disease can be bilateral. I recall one case in which it was necessary to ligate and divide the femoral vein in both legs due to the fact that the patient developed an embolus 5 days after the first leg was done. Dr. Donald McIntyre ligated this second vein during my absence on holidays. The patient made an excellent recovery.

It is likely unnecessary to point out that veins, like arteries, should never be ligated in continuity, but should always be divided. This will prevent any vasospastic phenomena resulting from ligation.

Summary

1. 85% of pulmonary emboli arise from the deep veins of the calf and sole of the foot.
2. Thrombophlebitis in these cases presents a clear cut picture in 75% of cases and such being the case, embolism can no longer be regarded as an unsuspected catastrophe for which there is no reliable prophylaxis and treatment.
3. Careful observation will reveal a large number of unsuspected cases of deep peripheral thrombophlebitis. We should be more embolus-conscious.
4. If the disease is present and no embolus has occurred, use:
   a. Homan's treatment;
   b. Heparin or coumarin;
   c. Ligate if this treatment fails.
5. If embolism has occurred, use heparin and ligate.
6. Thrombophlebitis in varicose veins accounts for 6% of post-operative pulmonary emboli. It is absolutely prevented by pre-operative saphenous ligation.
7. Femoro-iliac thrombophlebitis is an outspoken disease but only accounts for 2% of emboli. Use Homan's treatment and Heparin. This disease has interesting vasospastic sequelae which are amenable to surgical treatment.
8. Prostatic or Uterine thrombophlebitis accounts for 6% of emboli, diagnosed by exclusion and treated by Homan's method and heparin.
9. Heparin is advisable in all cases, but the cost is prohibitive. Coumarin is a poor second best, but must be used in:
   a. femoro-iliac variety;
   b. prostatic or uterine variety;
   c. mesenteric variety;
   d. occasionally in bilateral spreading deep peripheral thrombophlebitis which fails to respond to Homan's treatment and before bilateral ligation is attempted.
10. Deep peripheral thrombophlebitis and embolism is also found in purely medical cases and is sometimes apparently idiopathic. I saw one case in consultation which to all intents followed dancing.
11. 1 person in 17 with deep peripheral thrombophlebitis develops fatal embolus, but only ⅓ of emboli are fatal.
12. Encourage post-operative Trendelenberg position because it causes a rapid current in the legs and avoid the Gatch bed (embolism bed) because the flexed knees encourage stasis in the calf and the flexed hips encourage stasis in the pelvis.
13. Do not ligate veins without division.
14. The application of leeches has been superceded by heparin or coumarin.
15. Although stasis is not the only factor involved in the production of thrombophlebitis, it is by far the most important factor in the propagation or growth of the clot and clot propagation is the important factor in the pulmonary embolism.

Editorials and Association Notes

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In Unity

At the recent C.M.A. meeting in Jasper there was considerable discussion regarding health insurance. The keynote of the discussion was that the value of the influence of the C.M.A. on the shaping of policy depended to a very vital extent on the solidarity of the profession as indicated by the strength of its membership.

If you have not sent in your dues, please do so at once.

If for any reason you cannot or do not care to belong to both the C.M.A. and the M.M.A., will you not at least send in your $10.00 membership for the M.M.A? A very interesting programme is in preparation for the meeting of the M.M.A.—Sept. 23rd to 25th, at the Fort Garry Hotel, and we hope you are planning to attend.

Let's demonstrate that Manitoba doctors are alive to the need for organized medicine by being able to report to the Convention as close to 100% membership as possible.

Remember C.M.A. and M.M.A. dues, $18.00. M.M.A. dues $10.00.

Notice to Health Officers

To the Health Officers of those Municipalities where Immunization, and examination of High School pupils, was not, or could not be, carried out in the early summer, it is suggested by the President of the Health Officers' Association that arrangements be made to do this work as soon as the schools start this fall.

The first Annual Meeting of the Manitoba Health Officers' Association will be held in Winnipeg, Sept. 22nd next. All Health Officers in the Province will be personally notified further of the details as to place of meeting, time, etc.

The programme will appear in next month's issue of the Review. One feature of it will be a Question and Answer session. Questions must be in by Sept. 15th. Owing to the Secretary resigning because of moving away, send your questions to the President, Geo. Clingan, M.D., Virden, Man., who will arrange for answers to them.

Also, during the year, each Health Officer was sent a copy of a proposed constitution. The Executive Committee has gone over it and will submit certain amendments. Look up your copy and come prepared to offer suggestions. And come prepared with some concrete suggestions regarding remuneration for Medical Health Officers.

Executive Meeting

There will be an Executive meeting of the Manitoba Medical Association on Monday, August 17th in the Medical Arts Club Rooms, at 8 p.m.

Dinner will be served at 6:30 p.m.

A full attendance is desired, as arrangements for the Annual Meeting will be discussed.

H. D. KITCHEN, President.

North West Medical Association

The North West Medical Association met at Minnedosa on July 8th with a good attendance. The lectures were interesting and instructive, and were enjoyed by all.

Dr. Clarke spoke on dental infections, Dr. Pierce on Encephalitis and the Mosquito, Dr. McDiarmid on Common Eye Conditions, and Dr. Clingan on Health Officers' programme.

The next meeting will be held at Russell, April 12th, 1943.

Sulfanilamide in Acute Nephritis

Williams, Longcope and Janeway (Am. J. M. Sc. 1942, 203, 157) gave approximately 3 Gm. sulfanilamide daily to 42 patients with acute nephritis. After the acute stage 1.5 Gm. was given daily until all evidence of the disease had disappeared. There was one death in the acute stage. Of 108 controls, twelve died in the acute stage. After two years 74% of the sulfanilamide patients and 52% of the controls were considered to have recovered completely.

Obituaries

Dr. Sam Rodin

After a long illness borne with the utmost fortitude, Dr. Sam. Rodin died in the Winnipeg General Hospital on June 9, in his 49th year. Graduating from the medical faculty of the University of Manitoba in 1915, he immediately enlisted in the Royal Canadian Army Medical Corps and served overseas with the rank of captain until the end of the first World War. From 1919, almost up to the time of his death, he practiced in Winnipeg, with an interlude in 1937, when he did post-graduate work in internal medicine at London, England. On the outbreak of the present war he served as medical examiner with the armed forces. He was a lecturer in medicine at the University of Manitoba, a member of the honorary attending staff of the Winnipeg General Hospital and of the executive of the College of Physicians and Surgeons of Manitoba. His affiliations were with the B'nai B'rith, the Masonic Order and Shaarey Zedek Synagogue, and he was a charter member of the General Monash Branch of the Canadian Legion. Integrity and courage were stamped on him indelibly.

Dr. Robert Moore Best

Dr. Robert Moore Best of Killarney died in the Brandon General Hospital on June 30 at the age of 64. Born in Ireland, he received his degree in medicine there and practiced in England and South Africa before coming to Winnipeg in 1911. Seven years later he removed to Killarney where he carried on practice and was active in the life of the community. He is survived by his widow, three daughters and three sons, one of whom is Dr. Brian D. Best of Winnipeg.

Dr. Clarence Currie Everson

Dr. Clarence Currie Everson, a former president of the College of Physicians and Surgeons of Manitoba, died in the Winnipeg General Hospital on July 10, 1942.

Born in Morden 57 years ago, he graduated in medicine from Manitoba Medical College and took post-graduate work in Chicago. He practiced in Morden for thirty years and took an active part in the life of the community. He was a member of several fraternal organizations. He was interested in tennis and curling. His widow, a son, and two daughters survive him.

Quiet and dignified in manner, and skilled in his profession, he gained the respect of all who knew him.

Personal Notes and Social News

Dr. A. H. Boon formerly of Birch River, Man., is now located at God's Lake, Man.

◈ ◈

Dr. W. J. Bennett, formerly of Sherridon, Man., has recently joined the R.C.A.M.C.

◈ ◈

Dr. and Mrs. James A. Porter have announced the arrival of a son, Robert Henry.

◈ ◈

Major G. S. Williams, R.C.A.M.C., has recently returned from overseas for posting to other duties in Canada.

◈ ◈

Dr. and Mrs. D. S. McEwan are being congratulated on the birth of a son on July 2nd at St. Boniface Hispital.

◈ ◈

Dr. J. E. Rose has joined the R.C.A.F. with the rank of Flight-Lieutenant. He will be leaving early in August for Ottawa.

◈ ◈

Dr. Ivy Frances Falardeau has been appointed acting captain R.C.A.M.C. and will serve with No. 10 Company in Winnipeg.

◈ ◈

Doctor Harry Williams, medical missionary to China for many years, has arrived home safely with his wife and two daughters.

◈ ◈

Congratulations are being received by Dr. and Mrs. K. I. Johnson of Gimli, Man., on the birth of a daughter (Linda) July 5th.

◈ ◈

Dr. Emma Adamson is spending a month with her husband, Squadron Leader J. D. Adamson, who is stationed at Regina, Sask.

◈ ◈

Captain M. T. Kobrinsky, R.C.A.M.C., recently spent a short furlough in Winnipeg, from his post at a military hospital in Newfoundland.

◈ ◈

Dr. Kay Borthwick-Leslie has been appointed to the rank of acting captain in the R.C.A.M.C., and will be stationed at Camp Shilo Military Hospital.

◈ ◈

Dr. E. Dwyer has been appointed regional medical officer for the Canadian National Railways, Western Region, with headquarters in Winnipeg.

◈ ◈

Major F. Hartley Smith, C.A.M.C., who went overseas in command of No. 1 Motor Ambulance Convoy, has returned to Canada to take a new post.

Dr. S. W. Baker of Ladysmith, B.C., was a recent visitor to Winnipeg, accompanied by his son and daughter. They were guests of Dr. Baker's mother.

◈ ◈

Major and Mrs. George H. Ryan, of Hamilton, Ont., formerly of Winnipeg, have announced the birth of a daughter (Anne Penderell) on July 5th, at Toronto General Hospital.

◈ ◈

Dr. John Mugan, son of Mrs. J. J. Mugan and the late Dr. Mugan of Winnipeg, was married June 2nd to Harriette, daughter of Mrs. Ross and the late Mr. George Ross of Weyburn, Sask.

◈ ◈

Captain Brock M. Fahrni (Overseas R.C.A.M.C.) and Mrs. Fahrni are receiving congratulations on the birth of a son (Christopher Brock) on July 10th at Winnipeg General Hospital.

◈ ◈

Lieutenant John Edward Hudson, R.C.A.M.C., son of Mr. and Mrs. E. S. Hudson of Hamiota, Man., was married July 10th to Dorothy McAlister, younger daughter of the Rev. and Mrs. P. T. Pilkey of Winnipeg.

◈ ◈

Surgeon-Lieutenant John Mulvin Parker, R.C.N.V.R., son of Mr. and Mrs. E. D. Parker of Winnipeg, was married to Margaret Elspeth, daughter of Mr. and Mrs. Ralph Messenger, of Bridgetown, N.S., on June 20th.

◈ ◈

Dr. and Mrs. R. W. McCharles celebrated their golden wedding anniversary on Wednesday, July 15th. To commemorate the occasion, they were entertained at dinner at the Manitoba Club by their son, Dr. M. R. McCharles and Mrs. McCharles.

◈ ◈

Dr. Duncan Kippen, son of Dr. and Mrs. Robert Kippen, of Newdale, Man., was married June 29th in Winnipeg, to Marguerite Dorothy, daughter of Mr. and Mrs. Earl Badger, of Moose Jaw, Sask. After the wedding trip they will take up residence in Winnipeg.

◈ ◈

Dr. Robert Matthews Ramsay of St. Paul, Minn., formerly of Winnipeg, was married at Superior, Wis. on July 12th, to Evelyn Luceile, daughter of the late Mr. and Mrs. O. Carlson of Superior, Wis. Dr. Ramsay is the son of Mr. and Mrs. Robert S. Ramsay of Winnipeg.

Department of Health and Public Welfare

COMMUNICABLE DISEASE REPORT

May 21 to June 17, 1942

MEASLES: Total 520—Winnipeg 218, St. Boniface 79, Kildonan East 42, Mossey River 20, St. Vital 19, Brandon 16, St. Paul East 11, Kildonan West 10, Deloraine Town 8, Tache 7, Montcalm 5, Portage la Prairie City 5, Emerson Town 4, Oakland 4, Fort Garry 3, Swan River Town 3, Unorganized 3, Cornwallis 2, Cypress South 2, Daly 2, Flin Flon 2, La Broquerie 2, Norfolk North 2, Portage la Prairie Rural 2, Ste. Anne 2, Tuxedo 2, Virden Town 2, Beausejour Town 1, Brokenhead 1, Cartier 1, Elton 1, Minitonas 1, Morden Town 1, Norfolk South 1, Sifton 1, Swan River Rural 1, St. James 1, St. Laurent 1, Transcona 1. Late reported: Winchester 8, Deloraine Town 7, Portage la Prairie Rural 6, St. Vital 6, Ste. Anne 1, Brandon, 1, Springfield 1, Unorganized 1.

MUMPS: Total 235—Portage la Prairie City 49, Winnipeg 49, St. Boniface 32, Brandon 26, Transcona 13, Tuxedo 8, Whitehead 5, Fort Garry 4, Portage la Prairie Rural 4, St. James 4, Cornwallis 3, Daly 3, St. Vital 3, Virden Town 3, Coldwell 2, Minnedosa Town 2, Souris Town 2, Unorganized 2, Beausejour Town 1, Louise 1, Rivers Town 1, Sifton 1, Stonewall Town 1, Strathclair 1, Winchester 1. Late Reported: Brandon 5, Transcona 4, Arthur 1, Daly 1, Norfolk South 1, Portage la Prairie City 1, Unorganized 1.

CHICKENPOX: Total 146—Winnipeg 86, Virden Town 13, Stonewall Town 9, St. Andrews 7, Unorganized 4, Kildonan East 3, Rockwood Village 3, Melita Town 2, Minitonas 2, Minnedosa Town 2, Norfolk North 2, St. Boniface 2, Arthur 1, Brenda 1, Dauphin Town 1, Glenwood 1, Kildonan West 1, St. James 1, St. Laurent 1, St. Vital 1, Transcona 1, Tuxedo 1. Late Reported: Unorganized 1.

SCARLET FEVER: Total 134—Winnipeg 60, Brandon 17, Cornwallis 11, Portage la Prairie City 10, Fort Garry 4, Turtle Mountain 4, Flin Flon 3, Oak Lake Town 3, St. Andrews 3, St. Boniface 3, Tuxedo 3, Miniota 1, Minitonas 1, Neepawa Town 1, Norfolk North 1, Rosedale 1, Shellmouth 1, Springfield 1, St. Francois 1, St. James 1, St. Vital 1, Unorganized 1. Late Reported: Fort Garry 1, Unorganized 1.

TUBERCULOSIS: Total 43—Winnipeg 15, Stanley 4, Unorganized 4, Brandon 2, Ethelbert 2, Portage la Prairie City 2, Assiniboia 1, Dufferin 1, Flin Flon 1, Grandview Rural 1, Hanover 1, Kildonan West 1, Norfolk South 1, Portage la Prairie Rural 1, Silver Creek 1, Springfield 1, St. Clements 1, St. Laurent 1, Transcona 1, Tuxedo 1.

GERMAN MEASLES: Total 34—Brandon 12, Portage la Prairie City 10, St. Vital 4, Fort Garry 3, Tuxedo 3, Dufferin 1, Whitehead 1.

WHOOPING COUGH: Total 24—Brandon 6, Souris Town 5, Gilbert Plains Rural 3, Flin Flon 1, Kildonan East 1, Transcona 1, Unorganized 1, Winnipeg 1. Late Reported: Flin Flon 4, Gilbert Plains Village 1.

DIPHTHERIA: Total 22—Winnipeg 12, Brooklands 3, St. Boniface 2, Birtle Rural 1, Kildonan West 1, St. Clements 1, Tuxedo 1. Late Reported: Whitemouth 1.

INFLUENZA: Total 10—Brandon 1. Late Reported: Brandon 2, Selkirk Town 1, Bifrost 1, Portage la Prairie City 1, St. Vital 1, Turtle Mountain 1, Kildonan East 1, Morton 1.

LOBAR PNEUMONIA: Total 10—Ste. Anne 2, Whitewater 2, St. Boniface 1, Tache 1, Unorganized 1. Late Reported: Hanover 1, Siglunes 1, St. Vital 1.

ERYSIPELAS: Total 7—Winnipeg 3, Minto 1, Portage la Prairie City 1, Selkirk Town 1, Springfield 1.

ANTERIOR POLIOMYELITIS: Total 6—Lorne 2, Grey 1, Miniota 1, Portage la Prairie City 1, Winnipeg 1.

ENCEPHALITIS: Total 4—Lorne 1, Norfolk North 1, Norfolk South 1, Portage la Prairie City 1.

MENINGOCOCCAL MENINGITIS: Total 3— Portage la Prairie City 1, Roblin Rural 1, Transcona 1.

TRACHOMA: Total 2—DeSalaberry 1. Late Reported: Portage la Prairie Rural 1.

TYPHOID FEVER: Total 1—Ste. Anne 1.

SEPTIC SORE THROAT: Total 1—Cornwallis 1.

DIPHTHERIA CARRIERS: Total 1—Brooklands 1.

VENEREAL DISEASE: Total 149—Gonorrhoea 94, Syphilis 55.

TREATY INDIANS: Total 59—Mumps 41, Tuberculosis 10, Lobar Pneumonia 2. Late Reported: Lobar Pneumonia 3, Influenza 3.

DEATHS FROM COMMUNICABLE DISEASES

May, 1942

URBAN—Cancer 52, Pneumonia Lobar 8, Pneumonia (other forms) 8, Tuberculosis 7, Syphilis 6, Hodgkin's Disease 2, Erysipelas 2, Diphtheria 1, Influenza 1, Whooping Cough 1, Gonococcus Infection 1. Other deaths under one year 22. Other deaths over one year 186. Stillbirths 24. Total 321.

RURAL—Cancer 31, Pneumonia Lobar 5, Pneumonia (other forms) 16, Tuberculosis 15, Influenza 4, Lethargic Encephalitis 2, Syphilis 2, Measles 1. Other deaths under one year 27. Other deaths over one year 173. Stillbirths 12. Total 288.

INDIANS—Tuberculosis 5, Pneumonia (other forms) 4, Influenza 1. Other deaths under one year 4. Other deaths over one year 3. Total 17.

DISEASES	Manitoba May 21-June 17	Ontario May 17-June 13	Saskatchewan May 17-June 13	Minnesota May 17-June 13	North Dakota May 17-June 13
Amebic Dysentery				6	
Anterior Poliomyelitis	6		2	4	
Meningococcal Meningitis	3	21	1	1	2
Chickenpox	145	1191	83	194	
Diphtheria	21	4	9	7	3
Erysipelas	7	10	12	1	
Influenza	1	10	10	1	5
Encephalitis	4			1	6
Measles	489	968	96	2062	119
German Measles	34	184	50		
Mumps	221	1623	608		
Scarlet Fever	132	652	130	183	33
Septic Sore Throat	1	11	1		
Tularemia				1	
Trachoma	1				
Tuberculosis	43	202	32	39	47
Typhoid Fever	1	6		3	2
Typh. Para-Typhoid		7	7	2	
Undulant Fever		6			
Whooping Cough	19	263	10	112	33
Gonorrhoea	74	515			26
Syphilis	39	458			15
Diphtheria Carriers	1				

Manitoba reports 21 cases of DIPHTHERIA! Doesn't that give us a black eye!

SCARLET FEVER, MEASLES and MUMPS are still quite prevalent.

TYPHOID FEVER—a few cases; Manitoba had one.

To date of writing, July 15th, 15 cases of POLIOMYELITIS have been reported in Manitoba in 1942. This is about the usual number for a non-epidemic year. If we are going to have many cases of it this year, they should be quite numerous by the time you receive this copy.

ENCEPHALITIS—only 9 cases have been reported January 1st to July 15th, 1942. Of these five have died. Several of them have not been very typical of our 1941 epidemic. At this time, one year ago, our epidemic was well started. Have you read the symposium in the June 1942 issue of the Canadian Public Health Journal?